

Alcoholic cardiac beriberi

The introduction of dietary changes by Takaki at the beginning of this century had as dramatic an effect in eradicating beriberi from the Japanese navy as had the juice of lemons and limes on the incidence of scurvy in the British navy just over 100 years before. Osler¹ had speculated on the infective versus dietary theories for both beriberi and scurvy, and Eijkman had shown that those who developed beriberi in Java while living mainly on polished rice improved when they were also given the polishings. From the latter Funk later isolated an amine, and it was this "vital amine" which was the first vitamin to be described.

Thiamine pyrophosphate is a coenzyme needed at several stages in the metabolism of carbohydrate. It is found in the husks of rice, the germ of other cereals, and nuts, liver, and yeast. The daily requirement is about 120 µg/MJ (or about 0.5 mg/1000 kcal)—an amount comfortably contained in a normal Western diet. Thiamine is not stored in the body, and the effects of its deficiency become apparent within a few weeks, particularly if the diet is high in carbohydrate. Wernicke's encephalopathy and the two classic forms of dry and wet beriberi reflect the respective dependence of the nervous system and heart on glucose metabolism. One form of beriberi is often accompanied by features of the other, and the considerable oedema of wet beriberi is often accompanied by peripheral neuritis. Both forms may be accompanied by loss of appetite and gastrointestinal symptoms, together with profound fatigue; in Sinhalese *beri* means "I cannot."

The only cause of thiamine deficiency likely to be encountered in Britain and the United States is chronic alcoholism. Even in chronic alcoholics, however, the thiamine deficiency diseases are generally considered to be rare, because most of them take enough thiamine-containing foods. A recent report from New Zealand is therefore of considerable interest, since Ikram and his colleagues saw and studied five patients in as many years with alcoholic cardiac beriberi.² They speculate that alcoholic cardiac beriberi may be considerably commoner than has been thought.

The traditional view about alcoholic heart disease, well reviewed by Portal,³ is that there are two separate types. Alcoholic cardiomyopathy is of uncertain cause but is probably due to a specific chronic effect of alcohol on the heart rather than to any dietary deficiency; atrial fibrillation is common, and the characteristic feature is left ventricular failure. Alcoholic beriberi is thought to be much rarer. One of the interesting suggestions of Ikram and his colleagues² is that the two types of alcoholic heart disease may occur together, a view that has been postulated by others.^{4 5}

The usual features of cardiac beriberi are vasodilatation and consequent high cardiac output. The mechanism is not entirely clear, but accumulation of pyruvic and lactic acids probably plays a part, though their concentrations in the blood are not always raised. Damage to sympathetic nerves may also contribute to the vasodilatation. The extent of oedema is not closely related to the severity of heart failure, and protein deficiency may be present and be a contributory cause.

The typical clinical features therefore occur in alcoholics taking an inadequate diet, particularly in beer drinkers, whose high carbohydrate intake increases the demand for thiamine. Both anorexia and malaise may easily be attributed to alcoholism rather than to thiamine deficiency, and the features of peripheral neuritis are not always present. Pain in the chest is common and occurred in three out of five of the New Zealand

patients, all of whom had normal coronary arteriograms. A normal or near-normal electrocardiogram is common, but sometimes the T waves become inverted across the chest leads after thiamine treatment has been started. Non-specific but abnormal histological and electronmicroscopical changes were present in the cardiac biopsy specimens of all five New Zealand patients.

In classic cardiac beriberi in the East right heart failure was more common than left. In alcoholic cardiac beriberi as seen in the West, however, the pattern of left heart failure with dyspnoea, rales, and impaired left ventricular function is more common, so that the typical mode of presentation is biventricular failure with sinus rhythm. The diagnosis may be easily overlooked, especially if features of high cardiac output are absent, as they may be. At present the best diagnostic test for thiamine deficiency is measurement of the transketolase activity⁶ in red cells in vitro before and after the addition of thiamine pyrophosphate, but this test is not routinely available in most hospitals in Britain. Measurements of blood concentrations of pyruvic and lactic acids are unreliable as diagnostic guides. Ikram and his colleagues found the response to thiamine given during the course of cardiac catheterisation to be useful, but again this is not a test likely to be available in most hospitals where alcoholics with heart failure present.

The practical implications for clinicians are that they should remember the possibility of thiamine deficiency in patients with unexplained heart failure, particularly in alcoholics, and add thiamine to the conventional treatment. This condition may be more common in Britain than we have realised, particularly with the rising incidence of alcoholism.

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¹ Osler W. *The principles and practice of medicine*. 3rd edn. Edinburgh: Pentland, 1898.

² Ikram H, Maslowski AH, Smith BL, Nicholls MG. The haemodynamic, histopathological and hormonal features of alcoholic beriberi. *Q J Med* 1981;NS, 50:359-75.

³ Portal RW. Alcoholic heart disease. *Br Med J* 1981;283:1202-3.

⁴ McIntyre N, Stanley NN. Cardiac beriberi: two modes of presentation. *Br Med J* 1971;iii:567-9.

⁵ Robin E, Goldschlager N. Persistence of low cardiac output after relief of high output by thiamine in a case of alcoholic beriberi and cardiac myopathy. *Am Heart J* 1970;80:103-8.

⁶ Brin M. Erythrocyte transketolase in early thiamine deficiency. *Ann NY Acad Sci* 1962;98:528-41.

Toxicity of pertussis vaccine

"Contact your doctor or clinic for advice about vaccination," say the DHSS posters on whooping cough, challenging parents with the question "Is your baby protected?" What should we tell parents who ask for advice?

Public confidence in pertussis vaccine remains low: the acceptance rate of 80% before the adverse publicity in 1974 has fallen to 30% in recent years and is even below 10% in some parts of the country.¹ The large epidemic of whooping cough in 1977-80, with 32 deaths, had little influence on the vaccination rates, though the alarm to parents, the danger and damage to children, and the effort and expenditure on hospital treatment and home visits were real enough.^{1 2} With another epidemic persisting this spring complacency hardly seems likely to be the main cause of the continuing low levels of